This listing of claims will replace all prior versions and listings of claims in the application:

Listing of Claims

Claim 1. (currently amended) A transgenic, non-human mammal mouse comprising erythrocytes that produce a human hemoglobin, but fail to produce adult hemoglobin endogenous to said non-human mammal mouse.

Claim 2. (currently amended) The transgenic, non-human mammal mouse of claim 1, wherein said erythrocytes fail to produce non-adult hemoglobin endogenous to said non-human mammal mouse.

Claim 3. (cancelled)

Claim 4. (currently amended) The transgenic, non-human mammal mouse of claim 1, wherein said human hemoglobin is hemoglobin A.

Claim 5. (currently amended) The transgenic, non-human mammal mouse of claim 1, wherein said human hemoglobin is sickle hemoglobin.

Claim 6. (currently amended) The transgenic, non-human mammal mouse of claim 1, wherein said human hemoglobin is fetal hemoglobin.

Claim 7. (currently amended) transgenic, non-human mammal mouse of claim 1, wherein said human hemoglobin is an anti-sickling hemoglobin.

Claim 8. (currently amended) The transgenic, non-human mammal mouse of claim 7, wherein said anti-sickling hemoglobin is selected from the group consisting of Hb AS1, Hb AS2, Hb AS3, Hb AS4, and Hb AS5.

ATTORNEY DOCKET NO. 21085.0053U5 PATENT

Claim 9. (currently amended) The transgenic, non-human mammal mouse of claim 1, wherein said human hemoglobin is hemoglobin Kansas Porto Alegre.

Claim 10. (currently amended) The transgenic, non-human mammal mouse of claim 1, wherein said erythrocytes produce human fetal hemoglobin and human sickle hemoglobin.

Claim 11. (currently amended) The transgenic, non-human mammal mouse of claim 1, wherein precursors of said erythrocytes each comprise a human hemoglobin gene comprising a thalassemic mutation.

Claim 12. (currently amended) The transgenic, non-human mammal mouse of claim 11, wherein said precursors of said erythrocytes each further comprise a gene encoding a human γ -globin chain.

Claim 13. (currently amended) The transgenic, non-human mammal mouse of claim 11, wherein said precursors of said erythrocytes each further comprise a gene encoding a human β -globin chain.

Claim 14. (currently amended) The transgenic, non-human mammal mouse of claim 1, wherein said erythrocytes produce a human anti-sickling hemoglobin and human sickle hemoglobin.

Claim 15. (currently amended) transgenic, non-human mammal mouse of claim 1, wherein precursors of said erythrocytes each comprise a chromosome comprising a human γ -globin gene and a human .beta.-globin gene.

Claim 16. (currently amended) transgenic, non-human mammal mouse of claim 1, wherein precursors of said erythrocytes each comprise a first chromosome comprising a human γ -globin gene and a human β -globin gene, and a second chromosome comprising a human ϵ -globin gene, a human γ -globin gene, a human δ -globin gene, and a human β -globin gene.

Claim 17. (currently amended) The transgenic, non-human mammal mouse of claim 16, wherein said human β -globin gene encodes a β^s hemoglobin chain.

Claim 18. (currently amended) The transgenic, non-human mammal mouse of claim 16, wherein precursors of said erythrocytes each comprise a first chromosome comprising a human γ -globin gene and a human β -globin gene, and a second chromosome comprising a human ϵ -globin gene, two human γ -globin genes, a human $\psi\beta$ -globin gene, a human δ -globin gene, and a human β -globin gene.

Claim 19. (currently amended) A method of producing human hemoglobin, said method comprising expressing said human hemoglobin in the erythrocytes of a transgenic, non-human mammal mouse of claim 1.

Claim 20. (cancelled)

Claim 21. (currently amended) A method of testing a substance for efficacy in treating sickle cell anemia, said method comprising exposing a transgenic, non-human mammal mouse of claim 5 to said substance and monitoring a characteristic of sickle cell anemia in said transgenic, non-human mammal mouse following substance exposure, wherein amelioration of said characteristic of sickle cell anemia indicates a substance useful for treating sickle cell anemia.

Claim 22. (original) The method of claim 21, wherein precursors of said erythrocytes each comprise a first chromosome comprising a human γ -globin gene and a human β -globin gene, and a second chromosome comprising a human ϵ -globin gene, a human γ -globin gene, a human δ -globin gene, and a human β s globin gene.

Claim 23. (original) The method of claim 21, wherein said characteristic of sickle cell anemia is red blood cell sickling.

Claim 24. (cancelled)